Amendments to the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application:

1-16. (canceled)

17. (original) A method of inhibiting thrombosis, the method comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits formation of a thrombosis by the mammalian platelets.

18. (original) The method according to claim 17 wherein the mammalian platelets are human platelets.

19. (original) The method according to claim 18 wherein the PPARγ is human PPARγ.

20. (original) The method according to claim 17 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

21. (original) The method according to claim 17 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

22. (original) The method according to claim 17 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

23. (original) The method according to claim 17 further comprising:

administering the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

24. (original) The method according to claim 23 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

25. (original) The method according to claim 23 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

26. (original) The method according to claim 17 further comprising administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

27. (original) A method of treating or preventing a thrombotic condition or disorder, the method comprising:

contacting mammalian platelets, in an individual exhibiting symptoms of or predisposed to a thrombotic condition or disorder, with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said administering inhibits platelet activation to treat or prevent the thrombotic condition or disorder.

28. (original) The method according to claim 27 wherein the mammalian platelets are human platelets and the individual is a human.

29. (original) The method according to claim 28 wherein the PPARγ is human PPARγ.

30. (original) The method according to claim 27 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

31. (original) The method according to claim 27 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

32. (original) The method according to claim 27 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

33. (original) The method according to claim 27 further comprising:

administering PPARγ, the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to the individual in a manner that provides for said contacting.

34. (original) The method according to claim 33 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

35. (original) The method according to claim 33 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

36. (original) The method according to claim 27 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to the individual under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

37. (original) The method according to claim 27 wherein the thrombotic condition or disorder is selected from the group consisting of stroke, venous or arterial thrombosis, disseminated intravascular coagulation, myocardial infarction, pulmonary thrombo-embolism, and pulmonary hypertension.

38. (original) A method of improving the quality of a blood product, the method comprising:

providing PPARγ, a PPARγ agonist, an RXR agonist, an inducer of a PPARγ agonist, or a combination thereof; and

introducing PPARγ, the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof, to a blood product, wherein the PPARγ agonist,

the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof inhibits clotting or activation of platelets in the blood product and thereby improves the quality thereof.

39. (original) The method according to claim 38 wherein the blood product is selected from the group consisting of whole blood, plasma, concentrated platelets, or a white blood cell product.

40. (original) The method according to claim 38 wherein the blood product is a mammalian blood product.

41. (original) The method according to claim 40 wherein the mammalian blood product is a human blood product.

42. (original) The method according to claim 41 wherein the PPARγ is human PPARγ.

43. (original) The method according to claim 38 wherein said introducing is carried out prior to storage of the blood product.

44. (original) The method according to claim 38 wherein the blood product is whole blood and said introducing comprises:

collecting whole blood from a patient into a receptacle comprising the PPARγ agonist.

45. (original) The method according to claim 38 wherein the blood product is plasma or concentrated platelets and said introducing comprises:

collecting whole blood from a patient;

separating the plasma or concentrated platelets from the whole blood; and

combining the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof, with the plasma or concentrated platelets.

46. (original) The method according to claim 38 wherein the blood product is plasma or concentrated platelets and said introducing comprises:

collecting whole blood from a patient;

combining the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof, with the whole blood to form a treated mixture; and separating the plasma or concentrated platelets from the treated mixture.

47. (original) The method according to claim 38 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

48. (original) The method according to claim 38 wherein the RXR agonist is selected from the group of 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

49. (original) The method according to claim 38 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

50. (original) A stored blood product comprising:

a blood product that contains platelets and

an amount of PPARγ, a PPARγ agonist, an RXR agonist, an inducer of a PPARγ agonist, or a combination thereof that is effective to inhibit platelet activation.

51. (original) The stored blood product according to claim 50 further comprising an anticoagulant.

52. (original) The stored blood product according to claim 50 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

53. (original) The stored blood product according to claim 50 wherein the RXR agonist is selected from the group of 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

54. (original) The stored blood product according to claim 50 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

55. (original) The stored blood product according to claim 50 wherein the blood product is whole blood, plasma, concentrated platelets, or a white blood cell product.

56. (original) A method of inhibiting platelet aggregation comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits aggregation of the mammalian platelets.

57. (original) The method according to claim 56 wherein the mammalian platelets are human platelets.

58. (original) The method according to claim 57 wherein the PPARγ is human PPARγ.

59. (original) The method according to claim 56 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

60. (original) The method according to claim 56 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

61. (original) The method according to claim 56 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

62. (original) The method according to claim 56 further comprising:

administering the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

63. (original) The method according to claim 62 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

64. (original) The method according to claim 62 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

65. (original) The method according to claim 56 further comprising administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

66-105. (canceled)